FUNCTION AND RESPIRATORY RHYTHM IN OBESE PEOPLE

F. Liot, E. Bernier, D. Lemaigre,
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16. Abstract

The authors studied respiratory rhythm in obese patients. All obese patients have a low expiratory reserve. Obese patients suffering from chronic broncho-pneumopathy also have a high residual volume and a vital capacity reduction. Expiratory pauses often accompany their respiration similar to but still different from the Cheyne-Stokes syndrome.

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FUNCTION AND RESPIRATORY RHYTHM IN OBESE PEOPLE[1]

F. Liot, E. Bernier, D. Lemaigre,
J. -F. Dessanges and S. Poenaru[2]

Obesity has an effect upon respiratory function. Respiratory complications have a rather significant place in causes of death of obese people [22]. The major aspect of the respiratory attack is made up of an unusual Pickwickian syndrome connecting somnolence, alveolar hypoventilation and cardiac insufficiency [4]. We began the study of respiratory function in the obese with particular attention to the following points: respiratory volumes, arterial blood gas, and respiratory rhythm problems. We have tried to specify the differences observed according to whether the obesity was isolated or associated with chronic broncho-pneumopathy. /993*

Subjects Studied. Study Methods

Subjects studied. The obese people in this series were recruited from among the outpatients and hospitalized patients in the various services of the Cochin Hospital. The excess weight has been figured in percentage as a ratio of theoretic weight calculated according to Lorentz's formula:

Theoretical weight (in kg):

$$T - 100 - \frac{T - 150}{4}$$

where T represents the waist in centimeters.

For our study we took subjects with at least + 50%, corresponding to definite obesity. In only 5 cases did the excess weight exceed + 100%, with a maximum at + 150%.

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[1]Report presented at the 13 October 1972 meeting of the Medical Society of Paris Hospitals by C. Laroche.

[2]Central Functional Research Laboratory, Cochin Hospital (Department headed by Pr. J. Ualmejac), 27, rue du Faubourg Saint-Jacques, F75014 Paris

*Numbers in the margin indicate pagination in the foreign text.

The study includes 41 subjects divided into 28 cases of isolated obesity and 13 cases of obesity associated with chronic broncho-pneumopathy, diagnosed according to clinical data. The 41 subjects were divided into 17 men and 24 women. Among the cases of isolated obesity the average age was 51 (extreme ages: 76 and 16), in the mixed obesity group the average age was 64 (extreme ages: 79 and 38).

Study methods. 1. Determination of the pulmonary volume and rate was made with the help of a spirograph with oxygen compensation (Godart pulmotest). The functional residual capacity was determined in a closed cycle through dilution of a tracer: helium (Godart pulmo-analyzer). The values are expressed as percentages of theoretical values calculated according to Coates' scales.

2. The blood gases were determined either with arterial blood (femoral or radial) or with arterialized blood from the ear lobe after applying a vasodilator salve 10 to 15 minutes before sampling [5].

Measurement of the PaO_2 was made by the polarographic method (Clark's electrode). The $PaCo_2$ was determined by Astrup's interpolation method, and the pH was determined with a glass microelectrode.

Results

1. Ventilatory Volume and Rate

In the case of isolated obesity the static values of pulmonary volumes (Table 1) are close to normal (vital capacity, residual volume, total capacity). Only the volume of expiratory reserve is clearly diminished (Table 2). /994 On the average the expiratory reserve volume is lower than 500 ml, representing a reduction of more than half in proportion to normal value [2]. Since the vital capacity was close to normal, the reduction in expiratory reserve volume is most often associated with an increase in inspirational capacity.

The maximum ventilatory rates, and particularly the S.M.E.V., are somewhat diminished. The Tiffeneau ratio is slightly lower in proportion to the theoretical value.

In the case of obesity associated with chronic broncho-pneumopathy, the vital capacity is clearly reduced, the residual volume clearly increased, and the total capacity slightly greater than normal (Table 1). Here again the

expiratory reserve volume is clearly lower than normal (Table 2). There is a significant bronchial braking with a large reduction in the S.M.E.V. in proportion to its theoretical value, and Tiffeneau's ratio is clearly reduced.

TABLE 1. SPIROGRAPHIC DATA COLLECTED IN CASES OF ISOLATED OBESITY AND OBESITY ASSOCIATED WITH CHRONIC BRONCHO-PNEUMOPATHY (AVERAGE VALUES)

	Isolated Obesity (28 cases)	Obesity With Chronic Broncho-Pneumopathy (13 cases)	
Vital capacity	91.5 ± 7.5	72 ± 9.5	0.01
Residual volume	127.5 ± 12	167.5 ± 38	0.01
Total capacity	103.4 ± 5	112.5 ± 14.5	N.S.
S.M.E.V.*	81.5 ± 7	47 ± 9.5	0.001
V.C.**	89 ± 4.5	65.5 ± 11.5	0.001

*Secondary maximum expiratory volume
**Vital capacity

All values, including the ratio S.M.E.V./V.C., are expressed in percentages of the theoretical value. (Average values ± 2 standard errors.)

TABLE 2. STUDY OF THE RATIO E.R.V.*/V.C. IN OBESE PEOPLE IN NORMAL SUBJECTS

	$\frac{V.R.E.}{V.C.}$		
1. Normal subjects	31.5% ± 2		
2. Isolated obesity	15.5% ± 2.5	(1-2)	0.001
3. Obesity + Chronic broncho-pneumopathy	20.5% ± 3.5	(1-3)	0.001

*Expiratory reserve volume.

The difference between the two groups of patients studied is clear from a comparison of the figures (Table 1) and the diagrams of volumes and rates in the two groups (Figure 1).

2. Blood Gases

In the second group of obese subjects there is a clear reduction in PaO_2, while the $PaCo_2$ remains within normal limits (Table 3). If we compare the

two series of obese patients, there is no difference in regard to the average blood gas values. However, the distribution of $PaCO_2$ is not the same in the two series if we consider figures in detail [2]. Actually, nearly half of the obese subjects without any associated pathology have a $PaCO_2 \leq 35$ mm Hg, while the $PaCO_2$ is reduced in only one case in the other group. On the other hand /995
we know $PaCO_2 \geq 45$ mm Hg in only 6 out of 28 cases of isolated obesity, while we find that 5 times out of 13 $PaCo_2$ is elevated in the case of obesity associated with chronic broncho-pneumopathy.

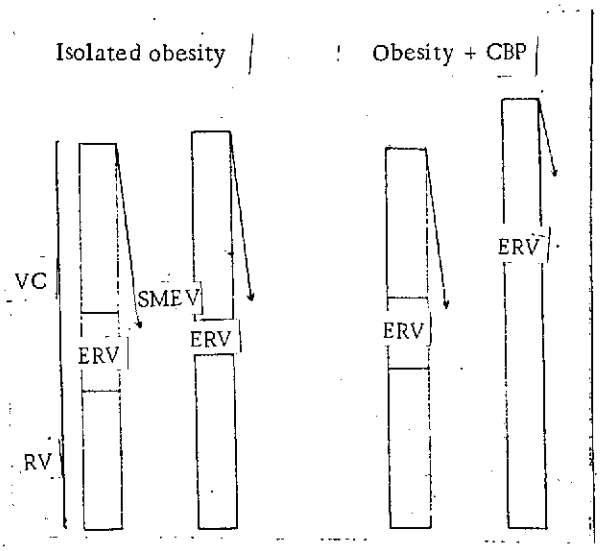

Figure 1. Spirographic Profile of the Obese Patient. Comparison of the populations involved with due consideration of age and sex. V.C., Vital capacity; R.V., Residual volume; E.R.V., Expiratory reserve volume; S.M.E.V., Secondary maximum expiratory volume.

TABLE 3. BLOOD GASES IN OBESE SUBJECTS

	Isolated Obesity	Obesity + C.B.P.
PaO_2 mm Hg	66.8 ± 4.5	64.6 ± 5.3
$PaCO_2$ mm Hg	38.5 ± 2.5	41.7 ± 3.5

3. Study of the Respiratory Rhythm.

In 21 out of 41 observations we noted anomalies in resting respiratory rhythm. In 15 cases this was a matter of irregularity in the current volume. In 6 cases it involved a typical periodic respiration.

Description of the anomalies. The simple irregularities in the rhythm seemed clearly different to us from the irregularities sometimes observed in nervous patients during spirography. They appeared in the 3rd or 4th minute of recording when the patients relaxed their attention. Five times out of 15 there was a rough pattern of periodicity. In a number of cases there were short pauses which did not exceed 6 seconds. Such anomalies were observed in the cases of isolated obesity and of obesity associated with chronic broncho--pneumopathy.

In 6 cases the anomalies appeared to be clearly periodic. They occur progressively after the branching on the spirographic cycle. In general pauses are seen at the end of an expiration lasting for several seconds, more extended expiration than real apnea. Between these phases there are periods of ventilation which can be construed as two different types:

a) inspiration following the pause is at first of normal value and the movements follow one another with the same amplitude (Figure 2 a). Sometimes the first movements of the ventilation cycle are fuller than the following ones (Figure 2 b).

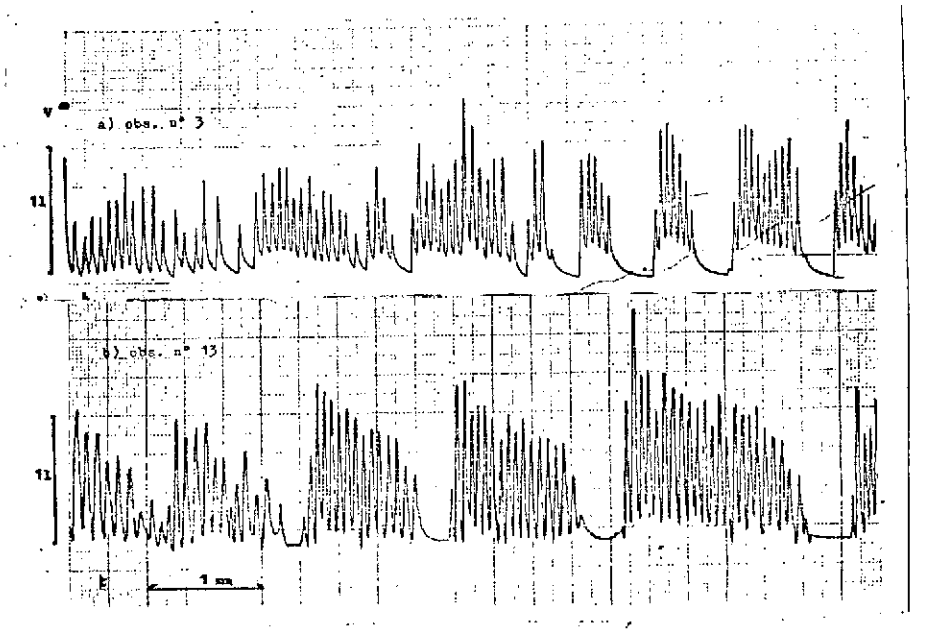

Figure 2. Periodic Respiration in the Obese Patient (see text).

b) there is an increase and a decrease in the current volume reminescent of Cheyne-Stokes respiration (Figure 3a). Nevertheless, there may exist a displacement of the current volume in the vital capacity which is unusual in Cheyne-Stokes respiration (Figure 3 b). In one case (Figure 3 c) ventilation looks like a succession of 3 or 4 respiratory movements with current volume displaced in the inspiratory reserve, followed by deep and extended expiration without real apnea. It should be noted that the total ventilatory rate is not modified greatly in this case between normal respiration ($\dot{V}$ = 11.385 l/min) and periodic respiration (10.720 l/min).

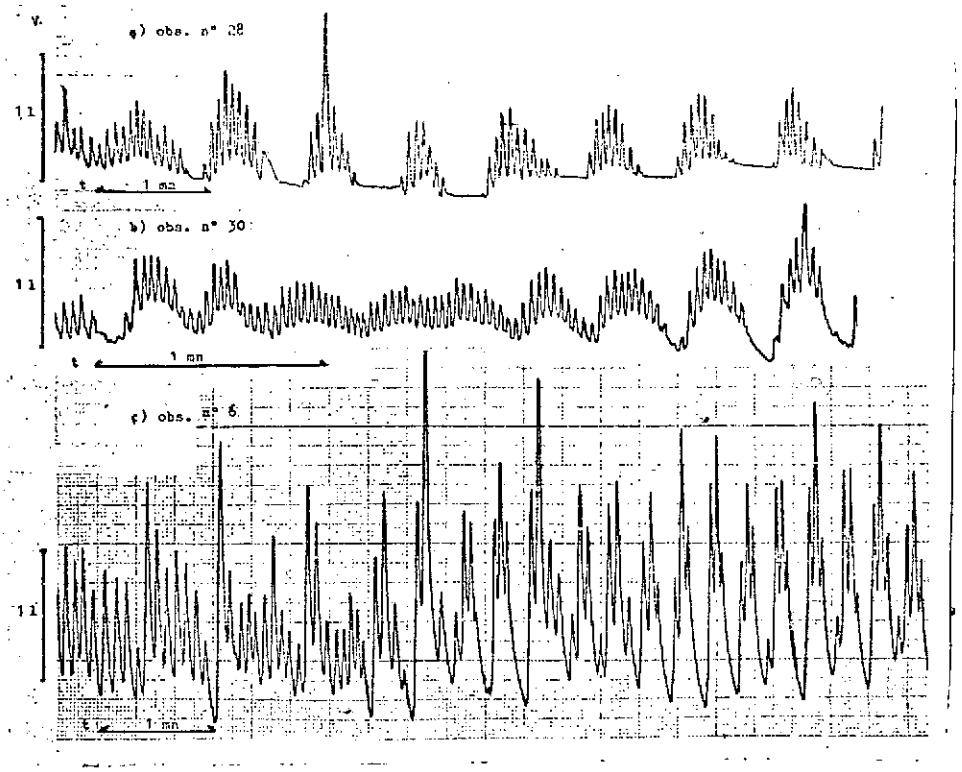

Figure 3. Periodic Respiration in the Obese Patient (see text)

Conditions of occurrence (Table 4). Periodic respiration has been observed in subjects from 49 to 76 years old. They have not observed it in younger patients. It has been noted 4 times in men and twice in women. It is more frequent in the case of isolated obesity than in association with chronic broncho-pneumopathy. /997

This usually involves obese patients with a considerable weight excess equal to or greater than 82%. In only 1 case of 6 was the obesity less, with weight excess reaching only 49%.

Definite sleep anomalies were not found in any of these cases. Only in 2 observations was there post-prandial somnolence, but there was no hypersomnia episode which could suggest the Pickwickian syndrome.

The $PaCO_2$ was elevated in 4 cases, normal in 1 case, and reduced in 1 case. In 4 cases out of 6 there was a significant drop in PaO_2 located between 50 and 33 mm Hg. In 4 cases out of 6 the hematocrit was elevated.

TABLE 4. PERIODIC RESPIRATION IN OBESE PATIENTS. CONDITIONS OF OCCURRENCE

No. of Observations	Age	Sex	Type of Obesity	Associated Clinical Signs	$PaCO_2$ mm Hg	PaO_2 mm Hg	Hematocrit %	Spirography
3	58	F	Isolated (+ 110%)	Post-prandial Somnolence	57	46	59	Normal
6	49	M	Isolated (+ 100%)	0	38	51	45	Obstructive insufficiency
10	51	M	Isolated (+ 84%)	Post-prandial Somnolence	52	53	58.5	Normal
13	68	M	Isolated (+ 82%)	0	48	65	60	Moderate restrictive insufficiency
28	76	M	Isolated (+ 49%)	0	32	63	44	Light mixed insufficiency
30	64	F	Mixed (+ 95%)	0	48	51	50.5	Obstructive insufficiency with distension

In most cases there was a moderate obstructive or restrictive insufficiency. In observations 3 and 10 sleeping and waking electroencephalograms were recorded. In observation 3 a sensitivity to hyperpnea was noted. On the sleeping recording there was not clear differentiation to be seen between the periods of somnolence and dozing, and the EEG sleep did not go beyond the second phase. Likewise there were a number of waking reactions caused by slight noises and by the deep inspirations following respiratory pauses.

In observation 10 a poorly organized waking EEG was noted. There were a number of "respiratory pauses", most often superimposable on periods of somnolence, accompanied by dull activities (1-4 c/sec., diffuse, of weak amplitude) ending by a renewal of the basic rhythm and of respiration at the same time.

In observations 13 and 30 waking electroencephalograms did not show any characteristic anomalies.

Discussion

1. The pulmonary volume anomalies do not call for much in the way of commentary. They confirm the generally held opinion that there are no significant respiratory volume anomalies in the case of isolated obesity except for the reduction in expiratory reserve volume [1, 7, 11, 15, 21]. Nevertheless, like others we observed a slightly increased residual volume [12, 24]. Should we, like B. Gamain et al., attribute this to some thoracic rigidity, or on the contrary to a modification in the bronchial closing volume explaining the reduction in E.R.V. and the increase in residual volume? [18].

On the other hand, when chronic broncho-pneumopathy is associated with obesity we find a clear picture of bronchial obstruction associated with a much more significant rise in residual volume and a reduction in vital capacity. The presence of such spirographic signs speak against the isolated nature of the obesity. In addition it has been pointed out that the cure of dieting in such cases has little influence on Tiffeneau's ratio [17].

2. Study of the blood gases brings out a major factor, reduction in PaO_2. When blood gas figures are collected, it is expedient to compare them

with the series of figures measured in normal subjects in the same laboratory and under the same conditions. The figures obtained in our laboratory in normal subjects, as a function of age, are given in Table 5. They agree with those of other authors [19, 25]. Furthermore, they confirm the validity of measurements made with arterialized blood taken from the ear lobe.

TABLE 5. PaO_2 AND $PaCO_2$ VALUES IN SUBJECTS SUPPOSED TO BE FREE OF CARDIO-RESPIRATORY AFFLICTIONS

Age	PaO_2 mm Hg	$PaCO_2$ mm Hg
< 30 years n = 36...	91 ± 3	38 ± 2
30-69 years n = 34...	83.4 ± 4	36.5 ± 2
> 70 years n = 30...	78 ± 3.5	36.2 ± 2

Such hypoxia in the absence of elevated $PaCO_2$ corresponds to an increase in the alveolo-arterial gradient. We can discard a diffusion problem. Each time that we have measured the capacity of alveolo-capillary transfer, we found it normal. The hypoxia is probably due to an increase in shunt effort in /998
connection with the inequalities in the ventilation-perfusion ratio [10, 16].

3. Difficulties in respiratory rhythm have already been pointed out during obesity [6, 10, 23]. Nevertheless their exact frequency has seldom been quantified and we were surprised to find them so often in obesity of the common type and outside of any cardio-respiratory decompensation: rhythm anomalies in 50% of the cases, and periodic respiration in almost 15% of the cases. B. Paramelle et al. point out 21 cases of respiratory pauses in 60 obese subjects [20]. It should be noted that in half of their cases the pauses were observed only when the patients breathed in oxygen-enriched gas.

On the semiological level the recordings are different from Cheyne-Stokes respiration, characterized by an increase and decrease in current volume follow lowed by apnea. Nevertheless, the spirograms which we are reporting, testify very well to a certain periodicity in anomalies, even if the periodicity is not perfectly regular. Only a critical study of the various kinds of difficulties in rhythm will permit classification and specification of the

morphological properties of these anomalies. The pauses observed always occur at the end of expiration. We have not observed any pauses occurring at the end of inspiration or at other moments in the ventilation cycle. The latter, which could be called anarchic pauses, have been pointed out but are certainly more unusual [20].

We have found these anomalies only in subjects reaching their 50's, whether the obesity is isolated or associated with chronic broncho-pneumopathy. None of them was affected by valvular cardiopathy nor by cardiac insufficiency. None of them had had a neurological episode suggesting cerebral arterio-scleroses.

We have just mentioned the classic causes of Cheyne-Stokes respiration. The latter is most often accompanied by alveolar hyperventilation with a clear drop in $PaCO_2$. For some people this is then a matter of apnea due to hypo-capnia [3]. We can eliminate such a cause for the beginning of the phenomena which we have observed. There is no drop in $PaCO_2$ except in one case out of 6. On the contrary the $PaCO_2$ is clearly elevated in 4 cases.

At the present time we do not have any explanation to offer. It has been maintained that the periods of apnea were not due to a suspension of the central respiratory activity, but to an obstruction of the pharyngo-laryngeal air passage [13, 14, 26]. Since the study of this series of obese patients we have undertaken simultaneous recording of esophagus pressures and air flow pressures. We have been able to observe both a persistence in intrathoracic pressure oscillations in their cessation along with the absence of respiratory flow in the same patient. This allows us to affirm that the physiopathological mechanism of respiratory pauses in the obese patient is not unequivocable [8]. On the other hand, this does not permit us to offer any total explanation of the relationships between obesity and respiratory difficulty anomalies. Only one fact remains: in general the anomalies disappear with dieting, showing the importance of treating obese patients. However, it can easily be conceived that the occurrence of significant pauses in very obese patients in cardio--respiratory insufficiency can have disasterous consequences because at that time in these patients the PaO_2 drops to critical rates at the same time as the hypercapnia [9] increases.

We cannot state that the difficulties we have observed are a minor phase of Pickwick's syndrome. Nevertheless such patients seem to suffer from a drop in awareness and from more facile drowziness. Perhaps it is an accentuation of these difficulties which leads to the hypersomnia found in certain cases.

Summary

Study of 41 cases of obesity, 13 of which were associated with chronic broncho-pneumopathy, permits the following conclusions to be drawn:

1. The pulmonary volumes are not very much modified in the case of isolated obesity, with the exception of the expiratory reserve volume which is always very diminished. When chronic broncho-pneumopathy is associated with obesity, there is, on the other hand, a clear obstructive syndrome with an elevation in residual volume and reduction in vital capacity. Here again the expiratory reserve volume is very reduced.

2. In all cases hypoxia is constant, without any cardio-respiratory insufficiency tendency. This hypoxia is most often associated with normocapnia. /999

3. Respiratory rhythm difficulties are present in half of the cases studied. In 15% of the cases they assume a rate of periodic respiration with expiratory pauses. This periodic respiration is accompanied by a tendency to drowziness and appears different from Cheyne-Stokes respiration.

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